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WITH INCONTINENCE OF THE VESICAL AND ANAL SPHINCTERS.

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BILATERAL CEREBRAL THROMBOSIS DUE TO SYPHILITIC ARTERITIS, WITH INCONTINENCE OF THE VESICAL AND ANAL SPHINCTERS.[1]

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I WISH to study carefully with you to-day the case of the man who is before us, as since his admission into the hospital it has presented several difficulties in regard to diagnosis. His history is as follows:

J. C., thirty-three years of age, a male laborer, of New York, but living in Colorado during the past three years, was admitted into this hospital July 11, 1893. His family history is unimportant, except for the occurrence of tuberculosis on his mother's side. His health was always good until four years ago, when he contracted a hard chancre, but without secondary manifestations. After this his health seems to have been fairly good until about one month prior to the occurrence of the paralysis for which he was brought to the hospital. The symptoms premonitory of the paralysis consisted of headache and mental dulness for one month, and, for a few days immediately antedating the attack, dizziness. On the morning of July 11th (the day of his admission into the hospital), while he was sitting in a chair, his left side became

[1] A clinical lecture delivered at the Arapahoe County Hospital.

paralyzed. He felt dizzy at the time, but he is quite sure that he did not lose consciousness. When he was brought to the hospital a few hours later, he seemed confused, but realized vaguely what was going on. His temperature was 98°, pulse 80, respirations 30, and there was incontinence of urine. He remained in about the same condition for four or five days, with normal temperature, total motor aphasia, and with complete paralysis on the left side, and nearly complete on the right side. At the end of this time he seemed brighter, and I had the following account of his condition recorded:

The mental condition seems to be good. The apparent mental dulness is partially caused by the motor aphasia, which is complete, as he is unable to utter the simplest monosyllable. The right leg is almost completely paralyzed. He is able to slightly flex and extend the right foot, but it is impossible for him to raise the foot from the bed. The flexors and extensors of the right knee and hip are extremely weak. Every muscle of the left leg is completely paralyzed and the knee-jerks are absent. The plantar reflexes on the right are slight, and for the great toe only, and on the left are absent. There is no ankle-clonus and the cremaster and abdominal reflexes are absent. The trunk muscles are weak, but not completely paralyzed, and the diaphragm acts well. The left arm and hand are entirely powerless; the right hand registers 48 on the dynamometer. The right-arm muscles are weak, but stronger than those of the leg. The deep reflexes are increased and most marked in the biceps muscle. The man has lost control of the sphincters of the bladder and anus. The muscles of the left side of the neck and face are paralyzed; those of the right side are weak. He is able to open and close the eyes and to frown. He can only protrude the tip of the tongue. Tactile sense, even over the external genitalia and around the anus, is well preserved. Although he seems a little slow in recognizing the contact of a substance,

this delay may be only apparent on account of his inability to talk. The senses of temperature, pain, posture, and localization are normal, but the pressure-sense seems a little deficient on the right side. Smell, taste, and hearing seem nearly normal.

The right pupil is regular and normal in size, and reacts to light and in accommodation; vision is good and the fields are preserved; there is no disturbance of any of the external ocular muscles; vessels, disc, and fundus present a normal appearance. The condition of the left eye is about the same as that found in the right eye. On account of the loss of control of the vesical and anal sphincters, the tactile sense was repeatedly tested over the external genitalia and around the anal region, but it was always found normal.

A few days after his admission into the hospital his temperature, which had before been normal, rose to 101°, and remained above normal four or five days, one evening registering 102°.

The condition of the man from the time of his admission to the present has shown a gradual improvement, especially in the power of walking. His speech is very much improved, and the side that was totally paralyzed has gained considerable power, but the bladder has never entirely regained its function. He is able to control the sphincter ani, and sometimes for several days at a time he is able to control the bladder; at other times he is totally unable to prevent the bladder from involuntarily emptying itself.

I will examine him again to day. He has fair power in the right leg, and uses it quite well. His gait is exceedingly awkward. The left leg is moved in a very ungainly manner, and in walking backward the left foot drags. He walks very much better forward than backward. The imperfect gait is due to muscular weakness and rigidity, and to lack of confidence in his ability to control his leg-muscles. You notice that his gait is different from that found in a person suffering from poste-

rior spinal sclerosis. Whilst there is some incoördination in his gait, it is of a jerky character and is due largely to increased muscular irritability, and to contractures that have taken place in the flexor muscles of the legs, especially on the left side. He experiences little difficulty in standing with his feet close to each other, and on closing his eyes the difficulty is only slightly increased. Turning around makes him decidedly dizzy. The right knee-jerk is greatly increased, the left still more increased. On the left side several contractions of the rectus femoris muscle occur from once tapping the patellar tendon—a rectus-clonus. Ankle-clonus is absent on the right; present on the left. There is increased irritability, both of the anterior and posterior tibial groups of muscles on the left side, causing "paradoxical" contraction of the tibialis anticus when the left foot is forcibly dorsally flexed. The plantar reflexes on the right side are absent on the inner side of the foot and are slight on the outer; on the left they are nearly the same. The cremaster and lower abdominal reflexes are absent. The epigastric reflex is decided on both sides. The deep reflexes of the arms are slightly increased on the right side, and on the left are decidedly increased. The muscular power in the right leg is fairly good; in the left, rather weak. The dynamometer on the right registers, 130; on the left, 112, so that muscular power is pretty good, even in the left hand, with which he was unable to move the register of the dynamometer at all when he entered the hospital. The muscles of the face show but little weakness. The tongue is protruded fairly well now, although for several weeks he was unable to get it far beyond the teeth. The pupils react to light and in accommodation, and are equal in size. The sensory functions, including the special senses, are all preserved. His speech is perfect now, but his mental power is slow.

I wish to study this case with you somewhat in detail, as it presents many interesting features, both as to the seat and nature of the lesion. With the exception of head-

ache and mental dulness, which antedated the paralysis for about a month, and dizziness, which immediately preceded the paralysis for only a few days, the man had always enjoyed good health, with the exception of syphilis four years ago, until the morning of July 11th, when, while sitting in his chair, without apparent shock or loss of consciousness, his entire left side, including the face, became completely paralyzed. Just when the right-sided partial paralysis came on he is not aware, but it probably was almost simultaneous in its onset with that of the left side, as both sides were affected on his admission into the hospital, a few hours after his attention was attracted by the paralysis of the left side. He had incontinence of urine and feces, and was unable to speak, although he was conscious.

The question naturally arises: Where are the lesions situated that would give rise to these symptoms? Are they in the brain, the cord, or the peripheral nerves, or are all these involved? The paralysis of the left side of the face, and the paresis of the right side, as shown by his inability to protrude the tongue, and the affection of speech, demonstrate conclusively that the brain is involved.

What evidences have we of a cord-lesion? In all cases attended with incontinence of urine and feces due to central nervous lesion, unassociated with loss of consciousness or dementia, we naturally expect to find the lesion in the spinal cord.

We can exclude myelitis in this case, because the tactile, pain, temperature, muscular, localization, and posture senses are all normal. Tactile sense has repeatedly been tested over the external genitalia and around the anal region, and at each examination it has been found normal.

In poliomyelitis there would be no incontinence of urine if the case were uncomplicated; and what is of more significance, in excluding poliomyelitis, we have the presence of increased myotatic irritability in muscles

hat were, a few weeks ago, absolutely paralyzed, and are at present paretic. The absence of muscular wasting and the presence of electric irritability to the faradic current also aid in excluding poliomyelitis, as well as myelitis.

Multiple neuritis of alcoholic origin is occasionally attended with incontinence of urine, and perhaps with an increased knee-jerk, while, possibly, ankle-clonus may be present in some cases.

This man has been addicted to the use of alcohol, but he never drank steadily or heavily. One of the chief features of multiple neuritis is its symmetry. From the start this case has been asymmetrical. There has been absolute paralysis of the left side, including the face, arm, and leg, with incomplete paralysis of the right side. Other points against multiple neuritis even of alcoholic origin are: involvement of the trunk and face-muscles; the affection of speech, with inability to protrude the tongue; the absence of the psychical disturbances so common in alcoholic multiple neuritis; and the maintenance of muscular nutrition, which is never found in any case of multiple neuritis, no matter what its cause may be. The paralysis of multiple neuritis never comes on suddenly, as it has in the present instance, and this disease is usually attended with sensory perversion, all symptoms being most marked at the distal portion of the extremities.

After a careful review of all the symptoms presented by our patient, both now and throughout his illness, I feel justified in excluding multiple neuritis and disease of the spinal cord. Our patient must be suffering from multiple brain-lesions which are bilateral. Will bilateral brain-lesions account for all the symptoms presented by this patient?

One hemisphere of the brain controls the muscles of the opposite side of the body concerned in movements of a higher or more differentiated character, and, to a certain extent, the muscles on both sides of the body,

which usually act in association with each other. The muscles that are most highly differentiated in their function from those of the opposite side of the body are those of the hand. The best types of bilaterally acting muscles are those of respiration, mastication, and those that move the eyes; therefore, a motor-paralyzing lesion of one side of the brain may cause absolute paralysis of the unilaterally-acting muscles of the opposite side of the body, and a slight weakness of the bilateral muscles of both sides. Such a lesion, unassociated with shock, unconsciousness, or mental disturbance of any kind, does not produce incontinence of urine or feces.

Sudden bilateral lesions of the brain occasionally occur, but they are usually unequal in their severity on the two sides of the brain. I see no reason for not attributing the incontinence of the sphincters of the bladder and anus to a bilateral brain-lesion.

Granting, then, that we have bilateral brain-lesions, it will be more convenient, in the present instance, to consider their location before trying to determine their nature. In all cases of sudden bilateral brain-lesions, our first thought is that the pons or medulla is implicated. The cranial nerves affected in our patient are the hypoglossal, making it impossible for him to protrude the tongue, and that portion of the facial, or seventh pair, distributed to the lower portion of the face. A sudden lesion in the nuclei of these nerves in the medulla must necessarily involve the nuclei of other cranial nerves, especially of the fifth, sixth, and eighth, adjacent to the facial nuclei, and of the ninth, tenth, and eleventh, which, are found close to the nuclei of the hypoglossal. Besides, such an extensive lesion must involve all the nuclei of the facial, thus affecting the upper as well as the lower portion of the face. Absolute paralysis of the same side of the face as of the body, with the upper portion of the face escaping, places the lesion above the middle of the pons. A lesion in the upper portion of the pons, causing paralysis of the opposite side of the body, will most

invariably affect the fifth nerve on the side of the lesion, and when the lesion is bilateral in this portion of the pons, sudden and extensive enough to give rise to the extensive paralysis found in this case, it seems impossible for the fifth nerve to escape. A lesion in the crura would affect the third nerves, so that we may conclude that the lesions that have caused the symptoms presented by this man, are situated in the cerebral hemispheres. In the cerebral hemispheres, bilateral lesions, or a unilateral one, giving rise to motor paralysis on both sides, or on one side of the body, as the case may be, may occur in the internal capsule, in the centrum ovale, or in the cortex. Sudden occlusion of the large branches of the middle meningeal arteries on each side of the brain, sufficient to give rise to bilateral paralysis of the face, arm, and leg, would be attended by convulsions, profound unconsciousness, marked disturbance in temperature, and probably speedy death. It is rare, even in unilateral sudden cortical lesions, that they are so extensive as to involve the whole of one side of the body. Lesions of the centrum ovale, causing bilateral paralysis, are exceedingly rare. When they occur near the cortex they give rise to symptoms simulating those of cortical lesions, and when near the basilar ganglia, symptoms very much like those of lesions occurring from injury to these ganglia. It is not always possible to differentiate them ; neither is it necessary for any practical purposes, except in a few rare instances. We must conclude, then, that the lesions in the patient whose case we are considering, are in the internal capsules, or in the white substance near them, but more probably in the former situation.

We shall next consider the probable nature of this man's lesions. For this purpose brain-lesions may be divided in regard to duration of onset into sudden, acute, and chronic. The chronic lesions that we need to consider to-day are morbid growths, aneurysm, and chronic abscess. The first two of these may be excluded by the

history and the absence of optic-nerve inflammation and resulting atrophy. Chronic abscess is excluded from the history and the absence of a cause. The acute lesions are inflammatory in their nature, are several days, or longer, in reaching their height, and are attended by a rise in temperature. The absence of temperature-elevation, and the sudden onset of the paralyzing lesions in the patient before us, enable us to exclude inflammation. The onset of our patient's trouble was sudden, and the lesions reached their height in from a few minutes to a few hours. All such lesions, excluding traumatism, are vascular in their nature.

The diagnosis then rests between embolism, hemorrhage, and thrombosis. In young subjects the chances are in favor of embolism, as against hemorrhage, with the presence of endocardial disease or syphilis, except when the apoplectic symptoms are severe, especially with prolonged and profound coma. The less the primary disturbance of temperature, in the majority of cases, the less likely is the lesion of a hemorrhagic nature.

It seems to me that in all cases of sudden and extensive paralyzing lesions of the brain, not attended with unconsciousness and deepening coma, and with but slight disturbance of bodily temperature, a hemorrhagic lesion can be excluded. Slight hemorrhages may occur suddenly, with but little variation of temperature; so also may large, gradually-increasing hemorrhages take place without great affection of consciousness or bodily temperature at first. This man's paralysis was extensive from the first; therefore, from the symptoms that he presented at the time of admission into the hospital, we are justified in excluding hemorrhage. The age of the patient is more in favor of occlusion of a cerebral vessel than of rupture, but we must bear in mind that the youth of a patient is never very strong evidence against cerebral hemorrhage.

Gowers (*International Medical Magazine*, October,

1893) implies that a hemorrhagic brain-lesion in the substance of the brain in the young is always fatal. This may be so, but I think I have seen at least one exception.

Having excluded hemorrhage, we have left to consider embolism and thrombosis. We are not justified in diagnosticating embolism unless we can find a source for the embolus outside of the cerebral circulation. In the vast majority of cases of embolism the embolus is caused by an old or recent endocarditis, a vegetation washed from the endocardium or from the valves of the cardiac orifices.

In our patient there is no history of cardiac trouble, and on careful auscultation no murmur is found over the heart. We must remember that it is possible to have slight vegetations attached to the cardiac orifices, and yet no murmur be discoverable. This is most likely to be met with several months after the acute cardiac inflammation. But, even in such cases, there is commonly a history of heart-trouble. In certain profound blood-changes there may be a tendency to coagulation of the blood in distant portions of the body, and a clot thus formed may reach the brain and occlude one of the main arteries. The only blood-change in the patient before us is due to syphilis, but this has not been very marked, as his color seems fairly good. Having failed to find a source of embolism we are justified in excluding it. There are, however, two other reasons for dismissing embolism: the bilateral character of the lesions in the cerebral hemispheres, and the preservation of consciousness at the time of the occurrence of such extensive paralysis.

Excepting in cases of involvement of the basilar artery, the brain symptoms from cerebral embolism are usually unilateral. When a vessel is occluded by an embolus, the occlusion takes place suddenly without premonitory symptoms, and if the vessel involved is a large one, consciousness is necessarily lost.

Having excluded the probability of hemorrhage or

embolism in this case, let us see if the indications of thrombosis are sufficiently well-marked to justify such a diagnosis. Thrombosis is generally preceded by distinct premonitory symptoms. These were fairly well-marked in our patient, and consisted of headache and mental dulness for a month or more, and dizziness for a few days immediately preceding the onset of the paralysis. The occurrence of extensive paralysis without loss of consciousness is rather in favor of thrombosis than of embolism. The rise of temperature that occurred in this patient several days after his admission into the hospital, usually occurs in most cases of extensive occlusion of the cerebral arteries, due either to embolism or thrombosis.

Having arrived at the diagnosis of cerebral thrombosis, let us extend our inquiries further, to determine, if possible, the nature of this condition. A thrombus may form in one of two ways: first, by coagulation of blood in the vessel at the point at which the thrombus forms, from profound blood-changes, as in cases of marked anemia, great blood-deterioration from the syphilitic virus, and from vitiated blood-states from various other causes; and secondly, from changes in the walls of the vessels, also at the site at which thrombotic occlusion takes place. In considering the subject of embolism, we found no reason to suspect an undue tendency in this man's blood to coagulation; therefore, we must attribute the cause of the thrombosis to changes in the arterial walls.

There are only two forms of disease of the arterial vessels that favor occlusion of the arteries that go to the great ganglia at the base of the brain. These are atheroma and syphilis. Atheroma is essentially a senile change, and probably rarely, if ever, occurs in a person as young as the man before us. We have, then, narrowed our diagnosis down to thrombotic occlusion of the arteries supplying the great ganglia at the base of the brain, and especially of those that carry blood to the anterior two-thirds of the posterior half of the internal capsules; and

this condition we have seen is due to syphilitic disease in the walls of these vessels. The changes that take place in vessels affected with syphilis consist in gummatous deposits, inflammation, swelling, and in consequence a narrowing of the caliber of the vessel at the point affected. Often the inner surface of the vessel at the seat of narrowing is rough, and thus the formation of a clot at this point is still further favored. The most marked changes take place in the larger arteries at the base of the brain, but the lessening of the caliber of the vessels is most pronounced in the smaller arteries given off from the main branches. Sometimes the narrowing goes on gradually, almost to the point of occlusion of the caliber of the vessel, but finally complete occlusion by the formation of a clot takes place suddenly.

If you bear in mind the essential difference between embolism and thrombosis, it will not be difficult for you to account for the difference in the onset of the symptoms in the two processes of arterial occlusion. In embolism, up to the time of the lodgment of the plug in the vessel, the cerebral circulation had been perfectly normal, and no premonitory symptoms precede the arterial occlusion. In thrombosis, on the other hand, the arterial blood-supply to a portion of the brain has been gradually lessening for weeks or months, and premonitory symptoms, in proportion to the diminished blood-supply, are the result. In some cases these are slight, and unless diligent inquiry into the previous health is made, will be entirely ignored by the patient; whilst in other cases they are pronounced, and often consist of temporary numbness, paresis, or paralysis, with or without aphasia. Headache, mental dulness, and dizziness are often well-marked, as they were in our patient.

The result of thrombotic occlusion of a vessel is necrotic softening of the part supplied by the occluded vessel. When a large vessel is occluded, extensive softening will take place unless the circulation to that

part of the brain is restored by collateral vessels. In the great ganglia at the base of the brain the nutrient arteries of these ganglia do not communicate with the arteries from the cortex, and in consequence, if the affected vessels are completely and permanently occluded, the softening will be complete, and the function of those areas of the directly-affected ganglia will be permanently lost. Fortunately, however, the vessels that are usually occluded in thrombosis at the base of the brain, in the neighborhood of the great ganglia, are often the smaller ones, so that the softening is not extensive. In all such cases the primary paralysis is more extensive than the permanent loss of power.

The prognosis in a case of thrombosis, other things being equal, is always worse when the trouble is bilateral than when it is unilateral. The immediate prognosis, or the chances of the patient escaping with his life, need not be considered here, as this man has passed this stage. What are the probabilities of his permanent recovery? It is unnecessary for me to go into all the elements that guide us in such an opinion, but let it suffice for the present for me to state that when ankle-clonus, exaggerated knee-jerk, and other evidences of increased myoatic irritability, with contractures of the flexor muscles of the extremities, are present, we have positive evidences of degeneration of the lateral columns of the cord, and neither a fair recovery nor marked improvement can take place.

The treatment of this case consists largely in making the man as comfortable as possible, encouraging him to go around on his crutches, but not allowing him to get fatigued; paying attention to the condition of the bladder, and trying to combat the affection of the arteries in order to prevent a recurrence of thrombosis.

In considering the treatment of syphilitic arteritis, it is necessary to bear in mind that the nodular deposits that take place chiefly in the outer and inner coats of the vessel are of slow and gradual growth, the caliber of the

vessel being narrowed more and more, until finally the vessel is occluded by clotting of the blood in the diseased vessel. The deposition in the walls of the artery is a specific process, and to a greater or less extent is influenced by anti-syphilitic treatment, but the clot that finally occludes the vessel is a non-specific process, and is but little, if at all, influenced by mercury and potassium iodid. When thrombosis occurs in a syphilitic subject, the thrombus and the resulting softening take the same course in spite of active, specific treatment, that we find to occur in occlusion of a vessel from atheroma; therefore the outlook, so far as the brain-injury is concerned, is no better than in the case of a similar injury due to embolism, hemorrhage, or atheromatous thrombosis. Please remember that the occluded vessel or vessels in a case of thrombosis due to syphilis is not the only vessel diseased, but that in all probability many of the vessels at the base of the brain are similarly affected, but to a slighter degree. Your course of treatment then must not be an expectant plan, but you should endeavor to prevent other vessels from becoming occluded. The time when anti-syphilitic treatment is most effective, both in relieving the patient from present symptoms and in averting a hopeless paralysis, is when the premonitory symptoms first manifest themselves.

In what must this treatment consist? In the administration of mercury and potassium iodid only? No. I believe that we often fail in accomplishing the object of our treatment in syphilitic cases by sole reliance upon these two agents. We must remember that most persons who are suffering from the chronic effects of syphilis are in an impoverished state of health; therefore it is often necessary to administer iron, quinin, arsenic, together with the most nutritious food, in order to get the desired effects of mercury and potassium iodid. I have seen syphilitic subjects who had failed to improve after weeks of active treatment by these agents, begin to feel better, and lose one symptom

after another on the same doses of potassium iodid and mercury that they had taken ineffectually for weeks before, after their general health had been improved by a tonic course of treatment. In syphilitic arteritis the large doses of potassium iodid that are so effectual in gummatous tumors of the brain are not only uncalled for, but they may be positively harmful by depressing the patient, impoverishing the blood, and thus favoring coagulation in the diseased arteries.